Permanent monocular blindness and ocular migraine

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Ocular or retinal migraine is an uncommon migraine variant that has an inconstant temporal relationship with headache. Transient visual loss with full recovery is the usual description and permanent sequelae are rare, but cases with visual loss have been described[1]. We report a case of ocular migraine that led to permanent blindness in one eye.

CASE HISTORY

A woman aged 48 attended the eye casualty with a 24-hour history of painless loss of vision in her right eye. She had had regular attacks of ocular migraine affecting her right eye since the age of 14. These had always begun with a central bright area with tiny sparks zigzagging across her vision. This became dark and spread within minutes to involve the entire field of vision in the right eye. Each attack lasted for up to one hour but always resolved spontaneously. She had stopped smoking after a myocardial infarction 2 years previously but had resumed after the death of her husband eight months ago. Her regular prescribed medication was aspirin 300 mg, atenolol 100 mg and frusemide 40 mg daily.

On examination her Snellen visual acuity was hand movements in the right eye and 6/5 in the left eye with full refractive correction. There was an afferent pupillary defect in the right eye. The ocular media were clear and the intra-ocular pressure was normal and equal in the two eyes. Fundus examination revealed a cherry-red spot at the right macula (Figure 1) and a normal retinal appearance in the left eye. No embolus could be seen anywhere in the arteriolar tree on fluorescein angiography (Figure 2) although arteriolar filling was abnormally delayed to 13 s (normal <10 s). All blood investigations, including erythrocyte sedimentation rate, full blood count and immunoglobulin screen, were normal. Gastric parietal cell antibodies were present at a titre of 1:80 but no other autoantibodies were detected. A carotid ultrasound examination was reported as showing some attenuation of the Doppler signal, particularly in the left carotid, but no evidence of flow disturbance. No mural thrombus was visible on cardiac ultrasound.

When she was reviewed three months after presentation her Snellen visual acuity was unchanged and the right optic nerve head had become pale and atrophic.

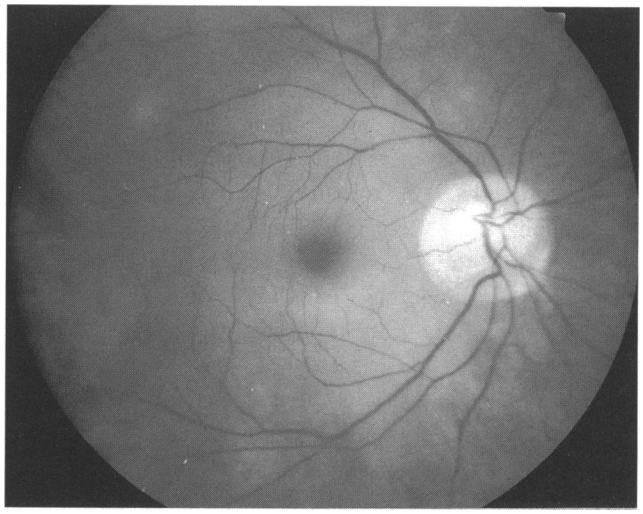

Figure 1 **Colour fundus photograph of right eye showing cherry-red spot at macula.** Note the calibre of the retinal arterioles. The disc appearance is typical of moderate myopia

COMMENT

Ocular migraine can be defined as a transient or permanent monocular visual disturbance accompanying a migraine attack or occurring in a person with a strong history of migrainous episodes[2]. The pathogenesis of arteriolar occlusion in ocular migraine is incompletely understood.

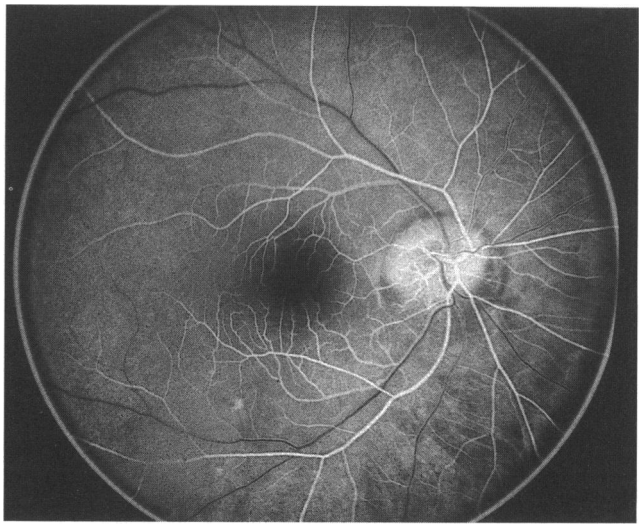

Figure 2 **Fluorescein angiogram of the right eye in early venous phase.** The whole arteriolar tree is filled completely with no evidence of embolus. The vessel bore is narrower than normal

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The most widely held view is that there is a transient reduction in blood flow due to vasospasm.

Migraine is strongly associated with ischaemic stroke in women, particularly amongst current heavy smokers (odds ratio 10.2) and amongst those using oral contraceptives (odds ratio 13.9)[3]. Smoking may promote platelet aggregation[4].

The retina can be thought of as part of the central nervous system and these odds ratios may be applicable here. Vascular complications such as myocardial infarction and central retinal vein thrombosis have been found to occur almost four times as often in patients with purely monocular visual symptoms in migraine as in patients with more classical migraine symptoms[5].

Systemic beta-adrenergic blocking agents have been used in migraine prophylaxis despite their peripheral vasoconstrictive side-effects, and the apparent absence of adrenergic receptors in the retinal arterioles[6]. The overall effect on ocular blood flow of reduction in systemic blood pressure and intraocular pressure has yet to be elucidated.

It is probable that patients with ocular migraine are at increased risk of developing permanent visual loss, especially if there are other risk factors for vascular complications. These have to be quantified but patients should certainly be warned to stop smoking.

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Giant oesophageal diverticula

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Less than 10% of oesophageal diverticula are intrathoracic[1]. Here they may become very large and surgical management can be complicated by the presence of more than one diverticulum.

CASE HISTORY

A man aged 75 reported a 2-year history of regurgitation with associated heartburn but no dysphagia. 2 years earlier, while being anaesthetized for investigation of a genitourinary complaint, he had had a cardiorespiratory arrest due to aspiration.

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Physical examination was unremarkable. A chest X-ray initially suggested the presence of a hiatus hernia (Figure 1). A subsequent barium meal revealed two large thoracic oesophageal diverticula, about 8 × 6 cm on the right and 6 × 4 cm on the left (Figure 2); reflux from the diverticula was observed. Endoscopic examination confirmed the presence of two wide-necked diverticula with no features of neoplastic change. On oesophageal manometry there was non-specific uncoordinated motility of the body. The lower oesophageal sphincter pressure profile could not be obtained because of poor access.

A laparoscopic transhiatal exploration of the diverticula was performed initially to explore the feasibility of resection; the necks of the diverticula were situated about 10 cm above the oesophagogastric junction. Later, both diverticula were resected with thoracoscopic assistance. Thoracoscopic mobilization and dissection of the diverticula was followed by a mini-thoracotomy (20 cm incision) to enable safe re-establishment of oesophageal continuity with interrupted 2'0 PDS vertical mattress sutures. Five days postoperatively a water-soluble contrast study of the oesophagus suggested a small anastomotic leak but a Gastrografin swallow at eleven days showed no leakage and the patient was discharged at fourteen days. Three weeks after operation, oesophagoscopy revealed a healed anastomosis with no compromise of the luminal diameter. The patient was symptom-free at six months follow-up.